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Anti obesity activity of *Coccinia Indica* in female rats fed with cafeteria and atherogenic diets

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ABSTRACT

Coccinia indica is a creeper locally available plant and upon literature survey revealed that it is used in conditions rheumatism, kidney problems, chronic bronchitis, arthritis and in obesity. Hence this present study was aimed to explore the anti obesity activity of fruit extracts i.e. alcoholic (ALFCI) and aqueous (AQFCI) in Cafeteria diet (CD) and Atherogenic diet (AD) induced obesity models in experimental animals i.e. female rats. Standard reference Sibutramine (5 mg/kg) produced a significant (P<0.01) anti obesity activity in CD and AD induced obesity in rats. Both ALFCI and AQFCI with medium (200 mg/kg) and high doses (400 mg/kg) exhibited a significant (P<0.01) anti obesity activity by reducing the body weight, food intake, organ and fat pads weight and serum GLU, CHO, TRG, LDL and VLDL cholesterol levels with an increased HDL levels in CD and AD induced obesity models in female rats.

Key words: *C. indica*, Seeds, Alcoholic and Aqueous extracts, Cafeteria and Atherogenic diet, Obesity, Sibutramine, Female rats.

INTRODUCTION

Obesity is a medical condition leading to reduced life expectancy with increased health problems because excess of body fat has accumulated to the extent that produce adverse effect on health. Body Mass Index (BMI), a measurement which compares weight and height, defines a person as overweight (pre–obese) when their BMI is between 25-30 kg/m^2, and obese when it is greater than 30 kg/m^2.[1]

Coccinia indica (Cucurbitaceae) is a creeper and most widely available in India. *C. indica* was reported for different medicinal uses i.e. used in rheumatism, kidney problem, chronic bronchitis, arthritis, obesity,[2] and it is already reported for anti diabetic and hepatoprotective activities. Hence the present study is an attempt to evaluate the anti obesity activity of *C. indica* fruit extracts in different models of experimental animals i.e. cafeteria (CD) and atherogenic diet (AD) induced obesity in rats.

MATERIALS AND METHODS

Cholesterol, Cholic acid, Lard oil from Sigma Aldrich, Bangalore, India; Sibutramine [Symed Laboratories, Hyderabad, India] and biochemical kits like serum GLU, CHO, TRG, HDL from Erba Diagnostics Mannheim GmbH, Germany; were used during the experimental study.

2.1. A. PREPARATION OF ALCOHOLIC EXTRACT:[3]

The dried *C. indica* fruit powder was packed in a soxhlet apparatus and extracted with 95% alcohol for 18 h. Appearance of colourless solvent in the siphon tube was taken as the termination of extraction. The extract was then transferred into a previously weighed empty beaker and evaporated to a thick paste on the water bath, maintained at 50^{o}C to get alcoholic (ALFCI) extract. The extract was finally air dried thoroughly to remove all traces of the solvent and the percentage yield was calculated with respect to the powder (g) used for the extraction.

B. PREPARATION OF AQUEOUS EXTRACT:[3]

About 100 g of *C. indica* fruit powder was taken into a round bottom flask (2000 ml) and macerated with 500 ml of distilled water with 10 ml of chloroform (preservative) for 7 days with occasional shaking for every hour in this vessel. Then the marc was removed by filtering the extract and it was concentrated on a water bath maintained at below 50^{o}C to get the aqueous extract (AQFCI) and percentage yield was calculated as mentioned above. Both the extracts were subjected to preliminary phytochemical investigations.

2.2 DETERMINATION OF ACUTE TOXICITY (LD_{50})[4]

The acute toxicity of ALFCI and AQFCI were determined by using female albino mice (16-25 g), maintained under standard husbandry conditions. The animals were fasted 3 h prior to the experiment and "Up and Down" (OECD guideline No. 425) method of CPCSEA were adopted for toxicity studies. Animals were administered with single doses of each extract and observed for their mortality during 48 h study period (short term toxicity). Based on the short-term profile of the extract next dose for the animals were determined. All the animals were observed for long term toxicity (7 days) and then 1/5th, 1/10th and 1/20th doses of the maximum dose tested 2000 mg/kg (maximal dose admissible) for LD_{50} studies were selected for the present study.

2.3: Determination Of Anti Obesity Activity:

Cafeteria Diet (CD) Induced Obesity Model In Rats[5]

The cafeteria diet consisted of three diets 48 g of condensed milk, 48 g of bread; 18 g of chocolate and 36 g of dried coconut and 48 g of cheese, 60 g of potatoes. The three diets were given to groups of 6 rats on days 1, 2 and 3 respectively and then repeated for 40 days in same succession in addition to normal pellet chow diet. The treatment for the experiment was protocol given below.

Experimental protocol

Group 1 - Normal control, which received normal pellet chow and water *ad libitum* (40 days)
Group 2 - CD control, which received CD + normal pellet Chow diet 40 days.
Group 3 - CD+ Sibutramine (5 mg/kg, p.o for 40 days)
Group 4 - CD+ Lower dose of ALFCI (100 mg/kg) p.o for 40 days
Group 5 - CD+ Medium dose of ALFCI (200 mg/kg) p.o for 40 days
Group 6 - CD+ Higher dose of ALFCI (400 mg/kg) p.o 4 for 0 days
Group 7 - CD+ Lower dose of AQFCI (100 mg/kg) p.o for 40 days
Group 8 - CD+ Medium dose of AQFCI (200 mg/kg) p.o for 40 days
Group 9 - CD+ Higher dose of AQFCI (400 mg/kg) p.o for 40 days

Atherogenic Diet (AD) Induced Obesity Model in Rats[5]

The AD consisted of 1% cholesterol (sigma)), 0.5%cholic acid (sigma) and 5% Lard oil (sigma) in addition to normal pellet chow diet. The treatment protocol for the experiment was given below.

Experimental protocol.

Group 1 - Normal control, which received normal pellet chow diet and water *ad libitum* (40 days)
Group 2 - AD control, which received AD + normal pellet Chow diet 40 days.
Group 3 - AD+ Sibutramine (5 mg/kg, p.o for 40 days)
Group 4 - AD+ Lower dose of ALFCI (100 mg/kg) p.o for 40 days
Group 5 - AD+ Medium dose of ALFCI (200 mg/kg) p.o for 40 days
Group 6 - AD+ Higher dose of ALFCI (400 mg/kg) p.o 4 for 0 days
Group 7 - AD+ Lower dose of AQFCI (100 mg/kg) p.o for 40 days
Group 8 - AD+ Medium dose of AQFCI (200 mg/kg) p.o for 40 days
Group 9 - AD+ Higher dose of AQFCI (400 mg/kg) p.o for 40 days

2.4. Statistical Analysis

Data generated from each experimental group animal studies n=6 were subjected for statistical analysis i.e. one way ANOVA followed by Dennett's't' test. P-values < 0.05^{+}, $0.01^{++}$and 0.001^{+++} were considered as statistically significant.

RESULTS

3.1. Preliminary phytochemical investigations

The ALFCI and AQFCI are subjected for preliminary phytochemical screening and both extracts found to contain carbohydrates, proteins, flavonoids, saponins, fixed oils, glycosides and steroids.

3.2. Toxicity study:

In the present study the both ALFCI and AQFCI are subjected for toxicity studies for the LD_{50} dose determination. Both the extracts when administered up to a maximum dose level of 2000 mg/kg body weight did not produce any mortality. Hence $1/20^{th}$ (low), $1/10^{th}$ (medium), $1/5^{th}$ (high) doses of the maximum dose tested for LD_{50} are selected for the present study.

3.3. ANTI OBESITY ACTIVITY:

Cafeteria diet (CD) induced obesity

Effect of ALFCI and AQFCI on body weight:

In normal control animals the body weight is noted as 172.21 g on 1^{st} day initial and 194.25 g on 40^{th} final day of the experimental study. A significant (P<0.01) increase in final body weight with 16.62 % increase (226.54) g is noted in CD induced obese rats. Sibutramine (5 mg/kg) significantly (P<0.01) reduced the final body weight with 11.53 % reduction (200.42 g) in CD induced obese rats. All the three doses of ALFCI reduced the final body weight low dose by 3.22 % (219.14 g) (P >0.05), medium dose by 6.21 % (212.46 g) (P >0.05) and high dose by 7.63 % reduction (209.2 g) (P<0.01) respectively.

All the three doses of AQFCI reduced the final body weight by low dose 4.49 % (216.35 g) (P >0.05), medium dose 6.28 % (211.05 g) (P >0.05) and high dose 8.97 % reduction of body weight (206.21 g) (P<0.01) respectively.

Effect of ALFCI and AQFCI on food intake:

In normal control animals the daily food intake is noted as 20.16 g/day. A significant increase (P < 0.01) in daily food intake is noted in CD induced obese rats as 30.28 g/day. Sibutramine (5 mg/kg) significantly (P < 0.01) reduced the daily food intake in CD induced obese rats i.e. 17.22 g/day. ALFCI and AQFCI with medium and high doses significantly (P < 0.01) reduced the daily food intake as 25.24, 22.01 g/day and 23.97, 21.90 g/day respectively. ALFCI and AQFCI with low doses also reduce the daily food intake i.e. 28.88 and 28.88 g/day (P >0.05) respectively.

Atherogenic diet induced obesity

Effect of ALFCI and AQFCI on body weight:

In normal control animals the body weight is noted as 172.21 g on 1 st initial day and 194.25 g on 40^{th} final day model. A significant (P<0.01) increase in final body weight with 20.84 % (234.75 g) is noted in AD induced obese rats as. Sibutramine (5 mg/kg) significantly (P<0.01) reduced the final body weight by 14.62 % (200.42 g) in this AD induced obese rats. All three doses of ALFCI reduced the final body weight by low dose 9.03 % (213.54 g) (P >0.05), medium dose 11.19 % (208.46 g) (P<0.01) and high dose 12.04 % (185.3 g) (P<0.01) respectively.

Similarly all three doses of AQFCI has reduced the final body weight as low dose by 8.87 % (214.16 g) (P >0.05), medium dose by 11.53 % (207.6 g) (P<0.01) and high dose body by 12.46 % (188.7 g) (P<0.01) respectively.

Effect of ALFCI and AQFCI on food intake:

In normal control animals the daily food intake is noted as 20.16 g/day. A significant increase (P < 0.01) in daily food intake is noted in AD induced obese rats as 30.37 g/day. Sibutramine (5 mg/kg) significantly (P < 0.01) reduced the daily food intake in AD induced obese rats i.e. 17.22g/day. ALFCI with medium and high doses significantly (P < 0.01) reduced the daily food intake noted as 25.24, 22.03 g/day. AQFCI also significantly reduced the daily food intake with medium and high doses i.e. 24.97, 21.94 (P < 0.01) and 24.09, 22.64 g/day (P < 0.01) respectively.

C). Effect of ALFCI and AQFCI on serum biochemical parameters

When compared to normal control group the serum biochemical parameters like GLU, CHO, TRG, LDL and VLDL except HDL levels were significantly higher in CD, AD induced obese rats. Sibutramine treated group exhibited a significant anti obesity activity by reducing all the biochemical parameters except HDL.

ALFCI and AQFCI significantly reduced the serum biochemical parameters like GLU, CHO, TRG, LDL and VLDL levels. Where in HDL levels are increased in a dose dependent manner in CD, AD induced obese groups (Table No 1-4).

D). Effect of ALFCI and AQFCI on organ and fat pads weights

When compared to normal control group the organ (Liver, Heart, Spleen, Kidneys) and fat pads weights (Mesentric, Uterus, Peri renal) were significantly higher in CD, AD induced obese groups. Sibutramine treated group exhibited a significantly reduced these organ and fat pads weights and exhibited an anti obesity activity.

ALFCI and AQFCI significantly reduced the organ and fat pads weights in a dose dependent manner in CD, AD induced obese groups (Table No 1-4).

Table No-1 Anti obesity effect of ALFCI and AQFCI on Serum biochemical parameters in Cafeteria Diet (CD) induced obesity in rats

Groups	Dose	Serum biochemical parameters (mg/dL)					
		GLU	CHO	TRG	HDL	LDL	VLDL
Normal Pellet diet	-	103.3 ±4.4	74.10 ±8. 0	94.2 ±6.9	20.19 ±1.3	35.05 ±8.8	18.85 ±1.3
CD Control	-	311.13 ±23.5**^{a}	184.55 ±20.28**^{a}	217.35 ±23.8**^{a}	14.0 ±0.5**^{a}	127.01 ±21.7**^{a}	43.47 ±4.7**^{a}
Stand	**SBT 5mg/kg**	110.7 ±4.7 **^{b}	102.29 ±7.79 **^{b}	122.78 ±5.1**^{b}	36.7 ±3.4**^{b}	40.99 ±9.0 **^{b}	24.55 ±1.0**^{b}
ALFCI	**100 mg/kg**	165.83 ±30.8 **^{b}	153.30 ±14.63 ^{ns}	159.33 ±8.2 *^{b}	16.1 ±0.45 ^{ns}	105.26 ±14.6 ^{ns}	31.86 ±1.6 *^{b}
ALFCI	**200 mg/kg**	111.12 ±4.8 **^{b}	132.73 ±12.0 ^{ns}	156.02 ±10.0 *^{b}	21.94 ±1.1 **^{b}	79.58 ±11.56 ^{ns}	31.20 ±2.0 *^{b}
ALFCI	**400 mg/kg**	104.75 ±4.4 **^{b}	120.44 ±23.55*^{b}	155.88 ±11.8 *^{b}	31.01 ±1.4**^{b}	58.24 ±19.0 *^{b}	31.17 ±2.3 *^{b}
AQFCI	**100 mg/kg**	176.98 ±18.9**^{b}	158.83 ±21.7 ^{ns}	164.85 ±18.7 *^{b}	15.85 ±0.46 ^{ns}	110.01 ±22.3 ^{ns}	32.97 ±3.7 *^{b}
AQFCI	**200 mg/kg**	116.86 ±14.4**^{b}	132.6 ±14.5 ^{ns}	160.0 ±9.7 *^{b}	21.72 ±1.2**^{b}	78.86 ±16.72 ^{ns}	32.01 ±1.9*^{b}
AQFCI	**400 mg/kg**	114.4 ±14.6**^{b}	118.3 ±3.2 *^{b}	142.42 ±9.7 **^{b}	27.03 ±1.0**^{b}	62.78 ±3.5 *^{b}	28.48 ±1.9 **^{b}

n = 6, Significant at P < 0.05, 0.01** and 0.001***, ns = not significant. a – compare to normal control, b – compare to CD control ALFCI – Alcoholic extract of fruits of C. indica, AQFCI- Aqueous extract of fruits of C. indica . SBT- Sibutramine. FP- Fat pad*

Table No-2 Anti obesity effect of ALFCI and AQFCI on Organ and Fat pad weights in Cafeteria Diet (CD) induced obesity in rats

Groups	Dose	Organ and Fat pad weights (g/100 g)							
		Liver	Heart	Kidney		Spleen	Peri-renal FP	Uterus FP	Mesentric FP
				Left	Right				
Normal Pellet diet	-	3.39 ±0.090	0.43 ±0.01	0.55 ±0.01	0.63 ±0.01	0.72 ±0.014	0.25 ±0.015	3.34 ±0.10	1.71 ±0.03
CD Control	-	4.75 ±0.52*^{a}	0.85 ±0.09**^{a}	0.77 ±0.01**^{a}	0.86 ±0.01**^{a}	1.23 ±0.07 **^{a}	0.59 ±0.014 **^{a}	5.26 ±0.02**^{a}	3.25 ±0.06**^{a}
Stand	**SBT 5mg/kg**	3.71 ±0.6**^{b}	0.51 ±0.03**^{b}	0.60 ±0.01**^{b}	0.58 ±0.01**^{b}	0.85 ±0.01 **^{b}	0.43 ±0.09 **^{b}	3.97 ±0.12**^{b}	2.09 ±0.08**^{b}
ALFCI	**100 mg/kg**	4.55 ±0.09^{ns}	0.75 ±0.2**^{b}	0.78 ±0.01**^{b}	0.78 ±0.01**^{b}	1.15 ±0.09 ^{ns}	0.52 ±0.017 *^{b}	4.85 ±0.05**^{b}	2.96 ±0.08**^{b}
ALFCI	**200 mg/kg**	4.22 ±0.08**^{b}	0.65 ±0.01** ^{b}	0.70 ±0.01**^{b}	0.70 ±0.04**^{b}	0.98 ±0.02 **^{b}	0.47 ±0.005 **^{b}	4.60 ±0.03**^{b}	2.63 ±0.06**^{b}
ALFCI	**400 mg/kg**	3.96 ±0.17**^{b}	0.55 ±0.01** ^{b}	0.63 ±0.07**^{b}	0.64 ±0.01**^{b}	0.86 ±0.07 **^{b}	0.44 ±0.09 **^{b}	4.27 ±0.05**^{b}	2.34 ±0.05**^{b}
AQFCI	**100 mg/kg**	4.51 ±0.10^{ns}	0.75 ±0.01**^{b}	0.72 ±0.07**^{b}	0.84 ±0.01**^{b}	1.19 ±0.11 ^{ns}	0.54 ±0.019 ^{ns}	4.94 ±0.06**^{b}	2.96 ±0.06**^{b}
AQFCI	**200 mg/kg**	4.17 ±0.10*^{b}	065 ±0.01 **^{b}	0.70 ±0.07**^{b}	0.72 ±0.01**^{b}	0.90 ±0.05 **^{b}	0.46 ±0.007 **^{b}	4.46 ±0.07**^{b}	2.61 ±0.07**^{b}
AQFCI	**400 mg/kg**	4.02 ±0.13**^{b}	0.54 ±0.01 **^{b}	0.65 ±0.01**^{b}	0.65 ±0.01**^{b}	0.84 ±0.01 **^{b}	0.44 ±0.011 **^{b}	4.30 ±0.05**^{b}	2.20 ±0.11**^{b}

n = 6, Significant at P < 0.05, 0.01** and 0.001***, ns = not significant. a – compare to normal control, b – compare to CD control ALFCI – Alcoholic extract of fruits of C. indica, AQFCI- Aqueous extract of fruits of C. indica . SBT- Sibutramine. FP- Fat pad*

Table No-3 Anti obesity effect of ALFCI and AQFCI on Serum biochemical parameters in Atherogenic Diet (AD) induced obesity in rats

Groups	Dose	Serum biochemical parameters (mg/dL)					
		GLU	CHO	TRG	HDL	LDL	VLDL
Normal Pellet diet	-	103.35 ±4.4	74.12 ±8. 0	94.25 ±6.9	20.19 ±1.3	18.85 ±1.3	35.05 ±8.8
CD Control	-	299.35 ±3.41^{**a}	232.44 ±9.76 ^{**a}	212.50 ±5.2 ^{**a}	12.6 ±2.4 ^{**a}	42.46 ±1.02 ^{**a}	224.29 ±22.38^{**a}
Stand	SBT 5mg/kg	125.90 ±1.41 ^{**b}	175.80 ±2.12 ^{**b}	139.70 ±1.04 ^{**b}	38.68 ±0.49^{**b}	27.94 ±0.20^{**b}	109.46 ±1.89 ^{**b}
ALFCI	100 mg/kg	258.03 ±6.41 ^{**b}	213.18 ±2.83 ^{*b}	205.87 ±3.74 ^{ns}	17.86 ±0.36^{**b}	41.17 ±0.30 ^{ns}	154.01 ±6.54 ^{**b}
ALFCI	200 mg/kg	186.87 ±1.56 ^{**b}	207.63 ± 2.63^{**b}	177.27 ±1.21 ^{**b}	23.2 ±1.17^{**b}	35.45 ±0.24^{**b}	148.98 ±3.28 ^{**b}
ALFCI	400 mg/kg	139.73 ±1.34^{**b}	185.77 ±1.17 ^{**b}	140.02 ±5.97 ^{**b}	28.43 ±1.71^{**b}	27.99 ±1.19 ^{**b}	128.51 ±2.44 ^{**b}
AQFCI	100 mg/kg	259.47 ±4.06 ^{**b}	219.07 ±1.967 ^{ns}	209.75 ±2.23 ^{*b}	15.13 ±0.64 ^{ns}	41.91 ±0.45 ^{ns}	158.98 ±3.70 ^{**b}
AQFCI	200 mg/kg	217.58 ±4.89^{**b}	205.47 ±3.34^{**b}	198.13 ±1.16 ^{**b}	24.80 ±0.77^{**b}	39.62 ±0.21 ^{*b}	141.04 ±2.97 ^{**b}
AQFCI	400 mg/kg	142.53 ±6.70^{**b}	181.50 ±3.46 ^{**b}	166.57 ±1.71^{**b}	28.5 ±1.09^{**b}	33.20 ±0.34^{**b}	104.26 ±1.33 ^{**b}

n = 6, Significant at P < 0.05, 0.01** and 0.001***, ns = not significant. a – compare to normal control, b – compare to CD control ALFCI – Alcoholic extract of fruits of C. indica, AQFCI- Aqueous extract of fruits of C. indica . SBT- Sibutramine. FP- Fat pad*

Table No-4 Anti obesity effect of ALFCI and AQFCI on Serum biochemical parameters in Atherogenic Diet (AD) induced obesity in rats

Groups	Dose	Organ and Fat pad weights (g/100 g)							
		Liver	Heart	Kidney		Spleen	Peri-renal FP	Uterus FP	Mesentric FP
				Left	Right				
Normal Pellet diet	-	3.39 ±0.09	0.43 ±0.01	0.63 ±0.010	0.55 ±0.013	0.72 ±0.014	0.25 ±0.015	3.34 ±0.10	1.71 ±0.03
CD Control	-	4.72 ±0.052**^{a}	0.85 ±0.09**^{a}	0.86 ±0.01**^{a}	0.77 ±0.01**^{a}	1.23 ±0.07**^{a}	0.59 ±0.014**^{a}	5.38 ±0.02**^{a}	3.53 ±0.06**^{a}
Stand	SBT 5mg/kg	3.69 ±0.06**^{b}	0.51 ±0.03**^{b}	0.58 ±0.02**^{b}	0.60 ±0.01**^{b}	0.85 ±0.01**^{b}	0.43 ±0.009**^{b}	3.97 ±0.12**^{b}	2.09 ±0.08**^{b}
ALFCI	100 mg/kg	4.51 ±0.10^{ns}	0.75 ±0.015*^{b}	0.78 ±0.01**^{b}	0.72 ±0.015^{ns}	1.19 ±0.11 ^{ns}	0.54 ±0.019 *^{b}	4.94 ±0.06**^{b}	2.96 ±0.06**^{b}
ALFCI	200 mg/kg	4.17 ±0.10**^{b}	0.65 ±0.010**^{b}	0.70 ±0.04**^{b}	0.70 ±0.01**^{b}	0.90 ±0.01**^{b}	0.46 ±0.007**^{b}	4.46 ±0.07**^{b}	2.61 ±0.07**^{b}
ALFCI	400 mg/kg	4.02 ±0.13**^{b}	0.54 ±0.011**^{b}	0.64 ±0.01**^{b}	0.63 ±0.07**^{b}	0.84 ±0.01**^{b}	0.44 ±0.011 **^{b}	4.30 ±0.05**^{b}	2.20 ±0.11**^{b}
AQFCI	100 mg/kg	4.55 ±0.09^{ns}	0.75 ±0.02**^{b}	0.84 ±0.09 ^{ns}	0.72 ±0.007^{ns}	1.15 ±0.09 ^{ns}	0.52 ±0.017**^{b}	4.85 ±0.05**^{b}	2.96 ±0.08**^{b}
AQFCI	200 mg/kg	4.22 ±0.08**^{b}	0.65 ±0.01 **^{b}	0.72 ±0.01**^{b}	0.70 ±0.05**^{b}	0.98 ±0.05**^{b}	0.47 ±0.005 **^{b}	4.60 ±0.03**^{b}	2.63 ±0.06**^{b}
AQFCI	400 mg/kg	3.96 ±0.17**^{b}	0.55 ±0.01**^{b}	0.65 ±0.01**^{b}	0.65 ±0.01**^{b}	0.86 ±0.07**^{b}	0.44 ±0.009**^{b}	4.27 ±0.05**^{b}	2.34 ±0.05**^{b}

n = 6, Significant at P < 0.05, 0.01** and 0.001***, ns = not significant. a – compare to normal control, b – compare to CD control ALFCI – Alcoholic extract of fruits of C. indica, AQFCI- Aqueous extract of fruits of C. indica . SBT- Sibutramine. FP- Fat pad*

DISCUSSION

Excessive energy rich food intake and lack of physical exercise leads to accumulation of body fat, the adipose tissue stores excess energy in the form of lipids, free fatty acid is liberated from lipoproteins by lipoprotein lipase and enters the adipocyte where it is reassembled into triglycerides. Cholesterol is a chemical compound that is naturally produced by the body and is a combination of lipid and steroid. About 80% of the body cholesterol is produced by the liver, while the rest comes from our diet. The liver is able to regulate cholesterol levels in the blood stream and can secrete cholesterol if it is needed by the body.

CD contains with a variety of highly palatable, energy rich, high carbohydrate foods elicited significant increase in body weights and fat pad mass in female rats. Cafeteria diets have been previously reported to increase energy intake and cause obesity in humans as well as animals[6].

Feeding animals with Atherogenic Diet (AD) has often been used to elevate serum or tissue cholesterol levels to study the etiology of hypercholesterolemia-related metabolic disturbances. Exogenous hypercholesterolemia causes fat deposition in the liver and depletion of the hepatocyte population; it can also cause malfunctioning of the liver,

which apparently follows micro vesicular steatosis due to the intracellular accumulation of lipids. In addition, feeding cholesterol rich diets induces free radical production followed by oxidative stress and hypercholesterolemia. Oxidative stress, which results from impairment of the equilibrium between production of free radicals and antioxidant defence systems, is one of the factors that link hypercholesterolemia with atherogenesis[7].

It is well known that hyperlipidemia is the leading risk factor for atherosclerosis. Epidemiological investigations revealed a positive correlation between the degree of severity of atherosclerosis and the concentrations of plasma cholesterol as well as LDL. Numerous population studies have linked raised concentration of total cholesterol or LDL–cholesterol in plasma with increased incidence of atherosclerotic events[8].

AD which contained high cholesterol and upon feeding to female rats a significant increase in different plasma lipids such as TC, TG and LDL with a parallel decrease in HDL was observed when compared with that of control group[8]. It was reported that consumption of CD and AD promotes obesity and fat accumulation in humans and several animal species, including rats, mice, and pigs[9].

Sibutramine is a serotonin–noradrenaline reuptake inhibitor (SNRI). A reuptake inhibitor inhibits the neuronal uptake of neurotransmitters and prolongs the duration of responses to both exogenous and neuronal release, in this case, of serotonin (5-HT) and noradrenaline (NA). Sibutramine has also been shown to block the reuptake of dopamine (DA) but at about a threefold lower potency when compared to 5-HT and NA. More recent studies suggest that sibutramine increases extracellular DA concentrations at similar levels to 5-HT in an animal model. 5-HT is recognised to have an influence on food intake and macronutrient selection.

Sibutramine, both extracts ALFCI and AQFCI had significantly ($P < 0.01$) reduced the physical parameters like body weight, food intake, organ and fat pads weight in CD and AD induced obesity models in female rats.

Sibutramine, ALFCI and AQFCI also significantly reduced the serum biochemical parameters like GLU, CHO, TRG, LDL-CHO, VLDL-CHO levels and increased the HDL levels in CD and AD induced obesity models in female rats.

CONCLUSION

Preliminary phytochemical investigations on both ALFCI and AQFCI were noted with carbohydrates, flavonoids, saponins, fixed oils, steroids, alkaloids and glycosides. No mortality or behavioural abnormality recorded in mice at the highest dose level of 2000 mg/kg tested for LD_{50} studies. Standard reference Sibitramine produced a significant anti obesity activity in the selected models from this studies it can be concluded that both ALSCV and AQSCV with medium and high doses exhibited a significant anti obesity activity by reducing body weight, food intake, organ and fat pads weight and serum GLU, CHO, TRG, LDL and VLDL cholesterol levels with an increase in HDL levels in CD and AD induced obesity models in rats.

The present study on anti obesity activity with ALFCI and AQFCI conformed because presence of several phytoconstituents like flavonoids, saponins, fixed oils, steroids, alkaloids and glycosides as these were already reported for their anti obesity activity.

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